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ON THE MORBID CONDITIONS OF THE KIDNEY, GIVING RISE TO ALBUMINURIA.

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[*Read before the Medical Society of London.*]

In the following communication, which I have the honour to lay before the Fellows of this Society, I purpose to present a sketch of the morbid conditions of the kidney which give rise to albuminous urine; to consider the essential nature of Bright's disease, so called; and the kind of treatment which pathology and experience recommend to us. So frequent is the malady, so often does it meet us in dealing with other diseases, so greatly does it mar our efforts for the cure of those diseases, and so fatal is it when arrived at its advanced stage, that no one, I think, can feel otherwise than deeply concerned to discover as much as possible of a cause so productive of evil. My descriptions of the structural lesions will principally be taken from my own examinations, which have been numerous; but I shall not fail to refer, as occasion may be, to the works of those who have thrown so much light upon the subject.

One or two brief remarks upon the physiological condition of the kidney may be premised. It has been well pointed out by Mr. Bowman, how peculiarly the vascular system of the organ is disposed; how the short and wide renal artery suddenly breaks up into small straight vessels, going direct into the Malpighian tufts; how the stream of blood thus arriving in those curious capillary loops, is thence checked and stayed, not having an ample, but, on the contrary, a rather narrow channel of egress by the efferent vessel, which passes into the tubular plexus. The effect of this arrangement is, of course, to bring a more than ordinary pressure to bear on the membrane of the capillaries: this, even in their healthiest state, must be greater than that exerted by the blood on any other capillaries; but if congestion arises, or the

engorgement stage of inflammation, this strain will doubtless be yet further and in a greater degree increased. The membranous wall of these capillaries (I am of course speaking of those of the Malpighian tufts) is strong and well marked; and it needs so to be, for these vessels, unlike any other that exist in the body, except perhaps those of the choroid plexus of the cerebral ventricles, are bare, not imbedded in the tissue they supply, and not supported by fibre or solid substance. How differently circumstanced are the capillaries of the liver; and how much rarer is hæmorrhagic effusion in this organ than in the kidney! Everything indicates that the natural and healthy function of the Malpighian capillaries is to effuse fluid; but fluid of what kind? Exact proof is perhaps impossible; and opinions are much divided. Mr. Bowman believes that water only is poured out from the Malpighian tufts; Mr. Simon, that a solution of albumen is effused; Ludwig, that the fluid is urine containing a large proportion of water, much of which is afterwards absorbed by the tubular venous plexus. Valentin holds the converse opinion, that the fluid from the tufts is at first very concentrated, and afterwards becomes diluted. I feel much inclined to adopt Mr. Bowman's view, because analogy makes it probable that the epithelium of the tubes is the agent in conducting the secretion of the solid matters; and experiments have shewn that animal membranes are capable of altering the composition of fluids that pass through them.

The condition of the kidney which is found in acute albuminuria, whether this be the result of the poison of scarlatina, or of any other cause, is not often seen in the dead-house. Frerichs mentions that in two hundred and ninety-two cases of fatal renal disease, there were only twenty that presented the appearances which characterise his *first stage*, that of hyperæmia, with commencing exudation. I have not seen it anything like so frequently, nor, indeed, should I expect to find it except rarely; as few of the cases of acute albuminuria prove fatal before the intensity of the congestive engorgement has been in a great degree removed by treatment, or has subsided. But when an opportunity does occur of inspecting a kidney in this state, it is found to present the following lesions. The size and weight are greatly increased, even to as much as twice the normal figure. This depends in great part on the extremely loaded state of the vessels, which are everywhere full of dark blood; the Malpighian capillaries, and those of the tubular plexus, are alike distended; as well as those distributed to the mucous membrane of the calyces and pelvis. When the congestion arrives at a certain point, extravasation takes place, and thus blood is poured out from the Malpighian capillaries into the capsules, and from thence down along the tubes; in other cases, from the venous plexus around the tubes; and again from the veins that run upon the outer surface of the organ.

The epithelial lining of the tubes is not apparently much altered; its function is doubtless more or less interfered with, partly in consequence of the congestion of the vessels, and partly from the plugging up of many of the tubules with coagulated fibrine. This substance I believe to be effused from the Malpighian capillaries in a fluid state, as liquor sanguinis more or less modified, to pass down the tubes for a variable distance, and to coagulate there, either by itself alone, or

mingled with various forms of epithelial matter, or together with these and blood globules. For a certain time, which varies according to circumstances, the fibrinous coagula remain where they have formed in the tubes; but, as the fluid accumulates behind them, they are forced on down the canal, and at last are swept out, and appear in the urine, whence they form, together with other matters, the well known red-brown sediment, so characteristic of acute albuminuria. The colour of the sediment is of course derived from the blood, the colouring matter of the globules exudes from them, and tinges the epithelial particles and the fibrinous casts themselves. I once had the opportunity of watching the formation of a sediment of this kind from its outset. A youth was in St. George's Hospital, under Dr. B. Jones's care, with symptoms partly of incipient phthisis, and partly of a more indefinite character, giving one the idea that he was hypochondriac or nervous. One day I observed his water to contain a copious dark reddish deposit, which was more distinct to the naked eye than to the microscope; this consisted of very delicate flocculi, which, under the microscope, appeared as extremely pale granular films entangling some renal epithelium. No albumen was present in the urine at this time; the next day there was a trace, and the deposit was more consistent, and some blood globules were observed with it. On the fifth day from its first appearance, the deposit was noted as of a still darker red, and heavier, consisting of amorpho-granular matter, large and numerous casts, and great quantities of renal epithelium. The urine was ascertained on the seventh day to be decidedly albuminous, but doubtless had been so several days before. There was pain at the lower part of the lumbar region; no dropsy. On the eighth day from the commencement, he was cupped on the loins, which had the effect of relieving remarkably the pain, and of diminishing the quantity of deposit and of albumen in the urine. He subsequently recovered in great measure, and quitted the hospital of his own will, or was discharged for irregularity, I am not sure which. In this instance, it seems that as soon as congestion reached a certain point, liquor sanguinis was effused from the Malpighian tufts; and, being but in small quantity at first, formed only those delicate pale films, which were afterwards replaced by the ordinary casts of the urinary tubuli.

It is a matter of great interest and importance to learn in what condition a kidney remains, which has been the seat of such a process as that we have just described. Is it probably seriously injured and deteriorated, or is it nearly restored, when the disease has passed by, to its pristine integrity? Doubtless this is a question to which a general reply can scarcely be given; but my own impression decidedly is, that in very many cases of acute albuminuria, such as occur in healthy children in consequence of scarlatina, or in adults from sudden arrest of perspiration, the kidney, under tolerably favourable circumstances, recovers completely. I may mention one specimen, which was given to me by Dr. Cotton; it was from a child who seemed to be recovering from dropsy consecutive to scarlatina. He was attacked, if I remember right, with serous effusion into both pleural cavities, and died. The kidney was large, pale, and wet; by far the greater part of its secreting structure appeared to be tolerably healthy, only here and there a

tube was seen which had completely lost its natural texture, and was evidently *hors de combat*![1]

When we consider how common a complaint scarlatinal dropsy is, and how many children recover perfectly from it, I think we may entertain good hopes that adults, with sound organs, and under proper treatment, may also recover perfectly, and with scarce any greater liability than others to the far more hopeless degeneration of the true morbus Brightii. I do not see why an engorged kidney should not be as completely restored as an engorged lung: nor do I think that the removal of the exudation matter is materially more difficult in the one than in the other.

The next anatomical condition which I propose to describe, is that of the large, more or less hard, and sometimes mottled kidney. This is considered by Frerichs to be the sequel of the first condition, which we have just considered; and in this opinion he is no doubt supported by others. Rokitansky, in particular, regards the mottled kidney as having its origin in a preceding hyperæmia. This point will be more properly considered when we have described the structural changes. The kidney is enlarged, its size and weight increased, even beyond that which occurs in the acute attack; it may rise as high as twelve or fifteen ounces. Its colour is pale, grayish or white, mottled or marbled, in various degrees, with streaks or patches of vascular redness; this applies to the cortical structure, the medullary usually presenting a contrast by its uniform and deep congestion. The capsule sometimes adheres closely, more often is easily separable from the surface. The tubes of the medullary cones seem to be separated from each other towards the base, and by their divergence produce an appearance not unlike a plume of feathers, or a sheaf of corn. On making a section of the cortical substance, the cut surface presents a peculiar confused aspect, not easy to describe, but such as one might conceive would be produced by a quantity of rather opaque matter coagulating in the interstices of a regular tissue. The surface is smooth, and presents nothing of a granular aspect. On microscopic examination, the cortical tubes are found more or less filled with altered epithelium; sometimes the quantity is so great as to obstruct and block up the tube; more often, I think, the canal is not entirely obstructed, but narrowed. Sometimes the epithelial particles are very perfectly formed and distinct; sometimes they seem to be lost in a dense stratum of granular matter. Often I have noticed that the epithelium has a peculiar *stiff* aspect,

[1] In another case of scarlatinal dropsy, where death took place after vomiting and convulsions, the kidneys were found pale, lobulated, and of healthy appearance to the naked eye. The microscope showed that the tubes, both of the cortical and medullary parts, were natural, except that a good many of them contained a reddish matter, which was probably altered blood. The Malpighian tufts appeared healthy. A long fibrinous coagulum was found in the superior longitudinal sinus, and also in the straight sinus; the former was larger in its middle than elsewhere, and discoloured and softened in this part. The brain was everywhere very "wet" and soft, the fornix softened so as to hang in shreds at one part, but the septum was not destroyed. The ventricles contained a pretty large quantity of turbid fluid; there was also a large amount of subarachnoid effusion, mingled at the base of the brain with a little lymph. These appearances may have resulted in part from the obstruction occasioned by the coagulum in the sinus; but for this, I think recovery might have taken place.

such as, compared with its natural condition, gives the idea of its vital actions or changes being arrested in part, or less readily carried on. A considerable quantity of oily matter in the form of minute drops is not unfrequently present in the epithelium; and when it is abundant, the kidneys have a decided milky white aspect. This oily matter, I think, is most often present in the medullary tubes near the base of the cones; it seems to be washed down from the cortical tubes, and to collect in this situation. In the interior of the tubes, there are often seen fibrinous casts, and sometimes globules of a yellow matter resembling very much those so frequent in the spleen, and which, like them, are probably derived from altered blood globules, or from effused hæmatin. The basement membrane of the tubes is often in great part lost; the tubes still retain perfectly their contour; but it is impossible to isolate a tube with its investing homogeneous sheath. This I think a very important circumstance, and one which seems to indicate clearly the influence of a degenerating process.

The Malpighian tufts are variously altered; some remain healthy, others are covered with films of coagulated fibrine, which obscure the capillary loops; sometimes the quantity of exudation is so great in the capsule as to compress the tuft, and reduce it into a small compass. The urine is mostly pale, contains a light-coloured sediment, consisting of casts, renal epithelium, and blood globules, in varying proportions. The foregoing description I wish to be very general, and to apply to all instances of enlarged kidney not decidedly hyperæmic, of confused or dense texture, and presenting under the microscope a more or less considerable alteration and increase of the epithelium lining the cortical tubes, without any actual destruction of them. Such a condition, I believe, may often ensue as the result of an acute attack of engorgement, which has been imperfectly subdued; but my impression is, certainly, that more often it is preceded by no decided hyperæmic stage, but that it arises insidiously, and proceeds without any noticeable disturbance of the system, until at last some secondary disease, the result of the unhealthy state induced in the blood, calls attention to itself, and perhaps to its latent cause. The fact to which Mr. Simon refers in his most instructive paper, in the *Medico-Chirurgical Transactions*, on subacute inflammation of the kidney, viz., that the mottled kidney is essentially scrofulous, confirms the view I have taken. Many of the cases I would include in the category just described, doubtless owe their origin to such an alteration of nutrition as may be properly termed subacute inflammation. In others, and I think the majority, the process is more purely and simply degenerative; while probably in all, at some period of their course, one form or other of morbid change predominates. This condition of kidney is very much more frequently found than that of acute engorgement; the number of instances observed by Frerichs amounted to a hundred and thirty-nine out of two hundred and ninety-two.[1]

[1] Although I have included in the above description all diseased kidneys presenting the specified characters, yet I think it most probable that they might be further distinguished into two classes, one of pale colour and mottled aspect, not of increased firmness in texture, and not having an adherent capsule; the other, not pale nor mottled, of decidedly increased consistence, and with its capsule almost invariably adherent. The former belongs more to the scrofulous diathesis, the latter to the fibrinous. Probably there are various intermediate grades.

The last general condition of the kidney which I propose to consider, is one which is perfectly familiar to us all, and which, from my own observation, I think is certainly the most common; that in which the organ is dwindled, contracted and granular on the surface. This Frerichs considers as the third stage in his arrangement; he calls it the stage of retrocession, of atrophy. The kidney, far from being enlarged, is in many cases exceedingly diminished, both in size and weight, sometimes descending so low as one and a half or two ounces. On the other hand, its consistence is greatly increased; it has often a leathery toughness, which is the more marked in the same ratio as the atrophy. The colour of the surface is more nearly that of the natural state than it was in the preceding condition; and this appears to depend on the presence of a greater quantity of blood. The capsule is always very adherent to the surface, sometimes scarce separable without laceration: it is not unfrequently thickened in radiated patches, and probably owing to this thickening may be split sometimes into two layers, a circumstance which I have seen to prove a cause of error, by leading to the opinion that the capsule, after its removal, had left a smooth surface, when in reality its deepest layer still remained adherent. The external form is often decidedly lobulated, reminding one in this retroceding condition of the fœtal kidney advancing in development. The granulations or prominences strewed over the surface are of a lighter colour than the intermediate tissue, and sometimes contrast with it very strongly; their size varies from that of a pin's head or poppy-seed, which is most common, to that of a hemp-seed or small pea. The wasting of the organ is especially manifested in the cortical structure, which is often reduced to a mere stratum a few lines in diameter; it also presents granulations in its deeper parts, similar to those on the surface. Wasting of the medullary cones also occurs, but to a less degree.

On microscopic examination of the cortical tubuli, nothing can be more marked than the utter atrophy by which they have been affected. In extreme cases, one may scarce find in a section anything except mere granular *débris*, some of these perhaps still preserving the contour of the original tube, but the greater part constituting an indefinite shapeless mass. In other less advanced cases, and in some parts indeed of all, the tubes are still discernible: they are irregularly distended and opaque, with granular contents, which have well-nigh, or perhaps completely, blocked-up their canals. Oily molecules, sometimes accumulated in considerable quantity, lie here and there among the granular matter, and increase the opacity greatly. The granulations are made up of the infareted convolutions of tubes, and seem to be the parts in which those traces of the natural structure still persist; they remain prominent, because the intervening parts, which are atrophied in a greater degree, have collapsed and shrunk in. Traces of basement membrane, according to my own observation, and that of my colleague, Dr. Sieveking, are generally indiscernible; it appears to perish and disappear, while yet the outlines of the tubes are tolerably preserved. In this respect, I differ entirely from Frerichs, who states that he still finds in the atrophied tissue the remains of the homogeneous membrane. This author also has found occasionally, in atrophied kidneys, new-formed fibrous tissue; he allows, however, that it is but rarely

present, and that its production is by no means the essential element of the disease. My observation quite accords with that of Mr. Simon, that the generation of new fibrous tissue in the kidney is somewhat doubtful, and that it has not much to do in occasioning the pervading atrophy.[1] The Malpighian tufts, in consequence of the general collapse, appear closer together; a few of them remain tolerably healthy, others are compressed and shrunken; often the capsule is filled to a greater or less extent with an oily looking material. I have not always been able to distinguish satisfactorily between the Malpighian bodies in this state, when their capillaries are greatly compressed, and the capsule partially filled with oily contents, and cysts which are so commonly found in kidneys thus affected. The tubuli of the medullary cones appear generally more affected by infaretion than by atrophy; they contain an altered, more coarsely granular, and hypertrophied epithelium, often mingled with considerable quantities of oily matter. I am inclined to think, that as the cortical structure, the normal seat of the secreting process, perishes, its function is in part taken up by the basal parts of the medullary cones which adjoin it; the medullary portion, at any rate, suffers far less from the atrophy than the cortical.

Thus far we have traced the atrophic changes of the original structure; but we have now to consider those peculiar formations which, at least by their form and number, appear to indicate the agency of an imperfect *vis reparatrix*, unable indeed to produce aught that can efficiently compensate for that which has perished, or which is capable of any higher development, but still, by its resemblance in exterior form to other natural parts, forcibly reminding the inquirer of that wonderful reparative power which the healthy organism so often displays. The cyst formation to which I allude, was, I believe, first correctly viewed and fully described by Mr. Simon, in his paper on subacute inflammation of the kidney. They had been often noticed before, and various opinions entertained of their origin, such as that they originated from dilatation of the Malpighian capsules, or from distension of the urinary tubules; but no one, I believe, had shewn in what great numbers they often existed, how minute was their original size, and how much of the apparent magnitude of a kidney might depend upon their presence. Since I first read his description, I have been in the constant habit of examining these curious formations; and, while well aware that other opinions respecting their origin were more generally held, I have never met with any evidence derived from my own observation that really militated against his view, but with much that tended to support it. Mr. Simon describes the cyst formation as a manifestation of new structure, as independent and superadded, though lowly organised growth. He believes each cyst to have its origin in a germ or nucleus particle, such as under healthy conditions might have produced an epithelial cell. This germ, instead of forming a nucleated cell, developes itself into a vesicle with clean sharp outlines, indicating the existence of a strong homogenous envelope. The cavity is

[1] Several observations, however, have inclined me to think that an *extra* tubular effusion of plasma does occasionally take place, which solidifies into granular matter, either alone or containing nuclei; once, also, I observed a considerable number of minute crystals, which seemed to lie between the tubes in the medullary and in parts of the cortical structure.

occupied sometimes by a clear fluid, sometimes by a granular matter, or by various admixtures of granular and oily matter. The size of the cyst varies extremely; the most minute are not larger than an epithelial particle, say $\frac{1}{1,000}$ inch, the largest may attain the magnitude of a cocoa-nut. This minuteness of the younger cysts seems to me, as it did to Mr. Simon, a conclusive argument in favour of the view above taken of their origin. If, as Drs. Johnson and Frerichs believe, they are dilated portions of tubes, how could they ever be seen of a magnitude inferior to that of the tubes themselves? The envelope of these cysts is occasionally of some thickness, appearing sometimes laminated, sometimes as if a second cyst were included within the first. It is rare that they possess a nucleus or any epithelial formation in their interior, but this I have certainly seen once; it seems to indicate a somewhat higher grade of development. The cysts are generally found in greatest numbers in the superficial structure of the cortical substance; and it is here that they attain, I think, their largest size, in the *same situation precisely* (be it observed) in which the degeneration of the natural structure attains its maximum. This, I think, is not destitute of significant import, in the view of their quasi-compensatory nature. Occasionally, however, they affect another situation, namely, at the bases of the medullary cones, just where these spread out into the cortex: when they are numerous in this part, I think they are generally absent from the surface. They are almost invariably closely impacted in the surrounding tissue, and are difficult to isolate completely; a circumstance much relied on by those who consider the cysts to be formed by portions of a tube being cut off by adhesion of its walls above and below a point, while the included part is distended into a pouch. That some cysts may not form in this way, I would not for a moment maintain; I think it quite possible that they may, and also from the Malpighian capsules, when the tube below is obliterated; but I do not think this is the mode of origin of the multitudinous growth which crowds so closely, in some specimens, the field of the microscope. One argument, which weighs very strongly with me in favour of Mr. Simon's view, is the fact that they are by no means found in all atrophied kidneys. Degeneration may have advanced to an extreme degree, without the production of any, or but a very few, cysts; on the other hand, numerous cysts may exist, without the tubes being in any great degree broken up: thus some other cause than the change taking place in the tubes seems requisite for the generation of cysts. The contents of a large cyst, which I recently examined, was a yellowish turbid fluid, abounding with large and small yellow-tinged granular corpuscles; in another, there were the same, with numerous free nuclei. A beautiful epithelial pavement, consisting of closely apposed nuclei, seemed to have constituted a lining to the larger. Dr. Bence Jones's researches into the chemical composition of the contents of these cysts, have shewn that they do not contain any of the organic principles of the urine, and thus that they are not, *quoad* function, compensatory structures. They contain aqueous and oily matter, and certain modifications of albumen. Mr. Simon, in two instances, detected a large proportion of xanthic oxide in them.

We come now to ask ourselves what is the essential nature of the morbid change, by which a healthy kidney is reduced to the condi-

tion we have last described: I am not now referring to the epiphenomenon of cyst production, but to the atrophic change itself. Is it essentially dependent on inflammation, acute, or subacute? on any condition of the part that necessarily is attended with pain, hyperæmia, exudation, or enlargement, and increased temperature? The language of most writers would imply at least such is the case. But ere the achromatic lens had begun to throw a new light on this obscure part of pathology, Dr. Prout, with that intuitive perception of truth which many of the fathers of medicine so eminently possessed, saw clearly, and announced distinctly, that the morbid process in Bright's disease was other and different from that belonging to any variety and type of inflammation. He called it, most justly, a *degeneration*, which might arise as the natural result of advanced age, or of premature decay from *innate inherited weakness* of the vital powers, or from *an acquired* weakness, as the preceding, either general or local.

All that has been ascertained respecting this disease, seems to me to confirm Dr. Prout's view. The textural changes, as they have been traced, seem to result from a slow, gradual change occurring in the renal tissue proper, not from alterations in the blood vessels, nor in the connecting tissue. There is but little, seldom any, trace of unequivocal products of inflammation; certainly they are not the prime cause of the morbid change. The very frequent latency of the disease, and its unobserved gradual progress for a long time, also indicate its degenerative, rather than inflammatory, character. The same is attested by the various conditions, which are justly regarded as the exciting cause of this disease; they are all such as exert a depressing, debilitating influence on the general system. Abuse of spirituous liquors, long continued exposure to damp and cold, poor and insufficient nourishment, together with excessive and fatiguing labour, exhausting discharges, venereal excesses, the mercurial and syphilitic cachexiæ, are mentioned by Frerichs as the principal causes of what he regards as the chronic form of Bright's disease.[1] To my mind, neither subacute inflammation of the kidney, nor chronic desquamative nephritis, convey a just idea of the pathological condition which really constitutes Bright's disease, and which so often produces the contracted granular kidney. I have not found the blocking up of the tubes by their epithelium so constant and marked as to assure me that it is the cause of their destruction; the tubes have often appeared greatly infareted when there was no evidence that the function of the gland was materially impaired. If, as we know, the fibrinous moulds and masses of epithelium can be washed out of the tubes for some time, and yet the organ subsequently recover its healthy condition, why should obstruction take place to so great an extent as to be the chief cause of the succeeding atrophy? My belief is, that the essential cause of this morbid change, is the supply of unhealthy blood plasma to the gland; herewith a general deterioration takes place of all the most important textures. The coats of the blood vessels are probably altered, and allow fibrinous and albuminous fluids to transude through their mem-

[1] The bacony (speckig) condition of the spleen often coexists with some forms of Bright's disease, and seems sometimes to result from a similar cachexia; but no one would rank the alteration of the spleen as a lienitis.

branes. The epithelium of the tubes is altered; it is more bulky, coarser, and stiffer than natural, and taking healthy structure as a point of comparison, seems certainly less fitted for the discharge of its function. I would compare its condition to that of a thickened aortic or mitral valve, which has become more rigid, thicker, and less capable of free play. Until we know what is the nature of the function which the epithelium fulfils, we cannot pronounce exactly in what its morbid change consists; but I am strongly inclined to think that it is not as a mechanical obstruction that it disorders the action and brings about the decay of the kidney, but by becoming unfit for its own proper service in the work of secretion. The basement membrane of the tubes is altered, and, I believe, destroyed; and this change can be considered no other than a pure degeneration. That into a gland thus altered, blood, which at the same time is unnaturally fluid, either from the effect of the disease or from the impairment which it induces of the circulating organs, should flow in undue quantity, and distend the tubular venous plexus (especially when there is general congestion of the veins) cannot be regarded as otherwise than natural; nor can it be admitted as any proof that hyperæmia is any necessary element in the morbid change which has taken place. I have said that I believe an unhealthy condition of the blood plasma, and, therefore, of the blood, to be the essential cause of the renal degeneration we are considering: but can we make this statement more determinate? can we show at all precisely what is the nature of the degenerative process, as illustrated, either by the condition of the kidney or that of the blood? It has been supposed that the process was essentially one of fatty degeneration precisely analogous to the fatty degeneration of the liver; but if this were true, then every cat in London has Bright's disease, for rarely indeed does one see in any human kidney such a prodigious quantity of fat or oil as is invariably present in the kidneys of these animals. But this accumulation of oil induces no destruction of tissue, the basement membrane of the tubes is beautifully distinct, and there is no atrophy of the cortical structure. All that has occurred is simply the addition of a certain quantity of oil to the epithelium of the tubes, just as when a person grows fat a certain quantity of oil is added in the form of adipose tissue to his body. The liver of most fish and of fatted sheep is very commonly gorged with oil, but it is not, therefore, in a degenerated condition. This, then, cannot be the essence of the change; but it is much easier to show what it is not than what it is. I have no theory to offer, but only two remarks. One is, that as alcohol exerts a well-known influence on albuminous matter, coagulating it when in a fluid state, and as we have frequent evidence of its influence in producing cirrhosis of the liver in persons addicted to its use, a disease which in very many instances seems to belong to the class of degenerations depending on an unhealthy condition of the plasma supplied by the blood; and as, moreover, we find that alcohol is a powerful agent in inducing the disease of the kidney we are now considering, it does not seem altogether a groundless idea that it actually produces, by its presence in the blood, the altered condition of the epithelium of the tubes, rendering it more bulky, stiff, and inapt to undergo the normal chemico-vital changes.

The other remark concerns a point to which my attention was long

ago directed by some writings of a German observer, I think Dr. Eichholz. He describes, if I remember aright, a condition of the general system, which might be termed the *fibrinous*, in opposition to the *tubercular diathesis*. In the former, there is a tendency to the formation of false membranes or serous surfaces, to the thickening and condensation of fibrous membranes, to such changes as produce thickened, stiff, puckered valves, white pericardial patches, cirrhosis of the liver, thickening of the capsule of the spleen, perhaps fibrous tumours in the uterus, and contracted and atrophied kidneys. The opposition between this diathesis of the tubercular, is well illustrated by cases of which I have seen a few, where old tubercular masses, or even single cavities, existed in the apices of the lungs, with surrounding induration and puckering of tissue, while some or other of the above-mentioned indications of the "fibrinous diathesis," were also present. The supervention of the tendency to fibroid formations arrested that to the deposit of tubercle. The above account is given from what I remember of Dr. Eichholz's statement, as I have not been able to find his paper again: I am pretty sure, however, that I have represented his views correctly, and my own observation has further inclined me to confide in their truth.

We will next proceed to consider the morbid changes which the blood and various organs of the body undergo in diseases connected with albuminuria. The blood drawn in acute anasarca, presents a buffy coat, often remarkably thick and firm. The serum, after the clot has separated, has been often observed of a turbid whitish or a milky aspect: this loss of transparency depends sometimes on the presence of oily, sometimes of albuminoid matter, sometimes on that of numerous colourless corpuscles. The essential urinary constituents are retained in the blood, and often give rise to the symptoms of toxæmia. In the true Bright's disease, the sp. gr. of the serum diminishes rapidly; from 1030, which may be considered its normal figure, it falls to as low as 1019 or '20; the solid constituents from 80 parts per 1000, fell to 60 or even 41·70: this change seems to occur more rapidly in proportion as dropsical effusions quickly take place. This diminution takes place chiefly at the expense of the albumen: the oily, extractive, and saline matters, do not differ much from the normal quantity; the latter, on the contrary, seem to be somewhat increased. The most important change is that which strikes the eye of the practitioner in the well-known sallow, whity complexion of the patient suffering from this disease: the proportion of red globules is remarkably diminished, sometimes to one-third of what it is found to be in health. The diminution proceeds throughout the whole disease, and seems to be even greater the longer the disease lasts. It might be supposed to be occasioned in part by the escape of blood globules from the Malpighian tufts into the urine; but I think it more likely that on account of the unhealthy condition of the liquor sanguinis, blood corpuscles are not developed in their proper amount. This is clearly a degeneration of one of the most important constituents of the blood. The quantity of urea and uric acid in the blood, seems to depend very much on the secretion being pretty free or otherwise; no proportionate relation can be shown to exist between the two substances.

Dropsy, in some form or other, is rarely absent in cases of true Bright's disease, at least during some period of its progress; out of 430 cases, there were 54 which presented none. The cause of its production has been variously assigned; some have considered it to result from a too dilute state of the blood; some from diminished pressure upon the walls of the vessels; some from alterations in the capillary vessels. I believe the first and the last of these three to be "veræ causæ," but that the latter is more essential; at the same time it is clear that failure of the heart's action or obstruction in any part of the circulation, will greatly tend to promote the dropsical effusion. That a dilute state of the blood is not the chief cause, is clear from those cases where dropsy is absent, although the disease has produced its other usual effects. Such patients are, I am convinced, exposed to great peril of sudden and fatal head attacks: they are struck down by apoplectic effusion, or often repeated epileptoid convulsions, die comatose, and on opening the body the perhaps latent cause of the evil is discovered in the spoiled and atrophied kidney. The cause of this increased peril doubtless is, that by the ordinary dropsical effusion much of the urea is carried off which otherwise accumulates in the blood, and produces its fatal effect on the brain.

The treatment of acute anasarca, whether occurring after exposure to cold and wet, after measles, or any other exciting cause, is sufficiently clear. The engorgement of the kidneys must be relieved, 1, by general blood-letting, if the patient's strength allow of it; 2, by cupping on the loins, which should never be omitted in any case, as it powerfully counteracts the local determination to the kidneys; 3, by tartar emetic, which, acting very much, as it does, in pneumonia, depresses the action of the heart, unloads the congested renal capillaries, promotes the action of the skin, and acts beneficially in diminishing the tendency to secondary inflammations. The employment of tartar emetic should be continued some time, the patient being of course kept in bed: warm or hot air baths should be occasionally employed, and this system persevered in until the urine is perfectly free from its dark sediment, gives no trace of albumen, and is passed in proper quantity. Many warnings have been given against the employment of saline diuretics in this complaint: I believe them to be as prejudicial while engorgement exists, as squills or the turpentines would be during the acute stage of a bronchitis or pneumonia; but in the later stages of the complaint, I am inclined to think in moderate doses they are beneficial. I have recently had a case under my care, in which, after the acute symptoms had been subdued by the treatment above mentioned, I gave several doses of nitre with a little nitric acid and a bitter. The urine became quite free from deposit, and almost so from albumen. I afterwards changed this plan for a chalybeate, giving the man small doses of the carbonate of iron, on account of his anæmic aspect; but I had soon to give this up, as it brought back again the turbid state of the urine, and had evidently induced fresh renal congestion. Again I returned to the nitre and acid, and the urine recovered its healthy condition, and continued so. The bowels should be kept open, but I should not recommend active purgation in this form of dropsical disease.

In the true Bright's disease, it is of much importance to entertain

correct ideas of the pathology; and I do not indeed think it altogether indifferent whether we call the disease a sub-acute inflammation or a degeneration. For if it be an inflammation at all, blood-letting naturally suggests itself as one of the primary remedies, and the inexperienced might readily commit the error of employing it when useless and injurious. But if we name the disease a degeneration, the mind at once turns to a more appropriate class of remedies. With respect to the secondary affections which renal degeneration induces, I can offer no new recommendations to the experienced Fellows of this Society, and these matters have been amply treated of by others. But whether anything may be done towards restoring the degenerated organ, and improving the quality of the deteriorated blood, is a subject scarce touched upon by any one, and yet of the highest interest. I can but offer a few suggestions. Of course when a kidney has utterly lost the greatest part of its cortical secreting structure, the lost tissue cannot be restored; we cannot create the organ anew. But we can never during life know for certain whether an organ *is* so wasted, or whether it is only in process of being so; we cannot tell whether it be not still possible to revive the decaying power, and to repair the injured mechanism. Our guiding ideas are, I think, two; one recognising the degenerative nature of the disease, its essentially low character, and therefore seeking to oppose it by all possible corroborative means, and such as increase the general vigour of the system. Cod-liver oil, iron, quinine, regulated and active exercise, a system of training, such as an athletic employs, mental occupation; these judiciously employed in the earliest stages would, I believe, have much efficacy in counteracting the degenerative tendency. There is no doubt that the phthisical tendency, nay, the deposition of tubercle, in the lungs, may be checked, and life greatly prolonged, by such measures; why should we not employ them, also, with effect in this kindred degeneration? One can hardly help feeling that our duty, as practitioners, is not confined to the treatment of disease as it arises and excites alarm, but where the known diathesis, the exterior tokens of the inward infirmity, inspire us with too good ground for future apprehensions, we should give timely warning, and show how far easier it may be to avert the coming of the evil while yet distant, than to arrest it when its course has once begun. The second idea which would guide my treatment is the following, and is founded very much on the peculiar condition of the epithelium of the renal tubule in the earlier stages of degeneration. We know that the alkalies and several of their salts exert a remarkable influence upon albumen and allied matters, dissolving them or tending to render them more fluid. Now it seems to me quite conceivable that alkalies, either pure, or preferably, perhaps, as vegetable salts, which become decomposed ere arriving at the kidneys, may so act on the bulky, coarse, heaped-up epithelium, as to cause it to assume a more fluid state, creating less obstruction, and more ready to undergo its appropriate changes. Such an effect, or at least a similar one, was certainly produced by Mr. Brandish in his treatment of scrofulous glands by liquor potassæ; and I cannot but think we may derive a useful hint from the practice he so successfully pursued. Of course during the trial of such a plan, care must be taken to uphold the strength adequately, and not to push the remedy too fast. If

this be not carefully observed, if the action of the remedy be not most carefully proportioned to the morbid process and to the general strength, it will cease to be a remedy; it will only act injuriously. I regret that I have not experience, to lay before the Society, of the result of such a plan of proceeding as I have traced. I have only employed it partially in one case; in this, indeed, all seemed to go on well for some time; the man, a gardener, who had Bright's disease in a marked form, kept to his employment, lost his dropsy, and seemed on the whole decidedly improved; but after some weeks the lungs became gorged and vomiting set in, which I was unable to restrain. Probably the renal degeneration was too far advanced, and the attendant circumstances were unfavourable; but still the results were sufficiently encouraging, to induce me certainly to try the plan again when a suitable opportunity may occur. Cupping on the loins even Frerichs does not counsel in Bright's disease, a measure which, if the disease depended essentially upon exudation into the kidney, would seem certainly advisable, at least in small quantities, or frequently repeated dry cupping. It should only be employed when there appears to be an attack of engorgement supervening on the degeneration, and then as sparingly as possible.

I fear I have already trespassed on the patience of the Society, and will conclude by thanking them for the kindness with which they have listened to me, and requesting that some of the members will favour us with their opinions as to the nature of Bright's disease, and the means they have found most successful in the treatment of it.

APPENDIX. I am inclined to think, from the condition of the urine in two cases which I have recently seen, that albuminuria may result simply from an altered and attenuated state of the membranous wall of the Malpighian capillaries. It is quite conceivable that this change in the vessels may have taken place without any coincident change in the epithelium; and indeed that this is possible, is proved by cases of so-called chylous urine, in which after death the kidney has been found perfectly healthy. The case recorded by Dr. Bence Jones supports the same view, as no traces of epithelium of the tubes were found in the urine: liquor sanguinis and blood globules were effused from the Malpighian tufts, but the epithelium was probably unaltered, as the normal solid constituents of the urine appear to have been present. The influence of exercise in producing the effusion of liquor sanguinis in this case, and of gallic acid in restraining and arresting it, also indicate that the disease is essentially of the nature of a flux taking place from the attenuated Malpighian capillaries, much in the same way as it might from the capillaries of a mucous surface. The point that arrested my attention in the cases I have mentioned, was the perfect transparency of the urine, and its freedom from any cloudiness or sediment; this, I think, is not usual in Bright's disease.